

of tapered stents to accommodate for the size mismatch between the ICA and the common carotid artery, may exert less expansile force on the carotid sinus.

In conclusion, patients with “significant baseline comorbid conditions” may be placed at a greater risk with CAS. It is currently unclear if CAS can compete successfully with CEA in a lower risk group. We are grateful to Ouriel et al for supplying us with this very interesting information.

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Reply

The points raised by Dr Kasirajan and colleagues are quite interesting and corroborate our own anecdotal findings. We, like they, have noted quite disturbing drops in heart rate and blood pressure during balloon dilatation at the carotid bulb. Interestingly, these changes do not appear to be as profound in patients with recurrent stenosis, presumably because the carotid sinus nerve has been disturbed at the time of carotid endarterectomy.

As Dr Kasirajan suggests, these hemodynamic changes are poorly tolerated by the very category of patients for whom carotid stenting might be appropriate: those patients with severe medical comorbidities. It is hopeful that the large randomized studies will help to sort out just the problems that have been identified by the Albuquerque group. Multivariate analyses of important clinical endpoints should shed light on exactly which patients are best served by carotid stenting or carotid endarterectomy.

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Regarding “High prevalence of mild hyperhomocysteinemia in patients with abdominal aortic aneurysm”

We read with interest the paper by Brunelli et al (*J Vasc Surg* 2000;32:531-6), as it exactly mirrors our own results published in abstract form. We have also studied homocysteine in patients with abdominal aortic aneurysm (AAA) coming to surgery and reported similar preliminary results in 14 AAA patients with 71% having homocysteine levels above the 95th percentile and mean homocysteine levels higher than in a comparable control group (17.8 vs 11.8 $\mu\text{mol/L}$; $P = .027$, Mann-Whitney).¹ Since that report we have recruited a further 46 patients for a total of 60 patients and recently reported homocysteine levels above the 95th percentile in 48% of the AAA patients (mean levels, 13.1 vs 10.9 $\mu\text{mol/L}$; $P = .003$, Mann-Whitney).² One interesting omission from the Brunelli paper was information on folate status and creatinine levels. These are both strong predictors of homocysteine levels,^{3,4} and a significant proportion of AAA patients will have some degree of renal impairment. Even mild renal impairment can lead to impaired clearance of homocysteine and raised plasma homocysteine levels. Did the authors examine folate and creatinine status in their aneurysm patients, and if so, did these variables influence their results?

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